Chronic Pain Management

An Overview of Taxonomy, Conditions Commonly Encountered, and Assessment

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KEYWORDS

• Chronic pain • Mechanisms of pain • Pain assessment

KEY POINTS

- Chronic pain has multiple mechanisms that result in pain amplification and maintenance, including central and peripheral sensitization and altered modulation of pain perception.
- Assessment of pain requires comprehensive assessment of symptoms and signs, suspected pain mechanisms, and patient's biopsychosocial context.
- Multiple validated measures exist for the assessment of pain symptoms, pain-related disability, psychological impact of pain, and candidacy for opioid management.

Chronic pain is a substantial health problem, both in its prevalence and its impact. The prevalence of chronic pain in a 12-month period has been estimated at 43% of the US population and 38% worldwide.[1] Institute for Health Metrics and Evaluation data rank low back pain seventh among global causes of disability-adjusted life-years, and, if combined, low back and neck pain would rank third.[2] Among US adults, the reported prevalence of chronic pain ranges from 2% to 40%, with a median of 15%.[3] In 2009, a National Health Interview Survey demonstrated that during a 3-month period, 16% of adults reported having a migraine or severe headache, 15% pain in the neck area, 28% pain in lower back, and 5% pain in the jaw or face.[3]

The impact and prevalence of chronic pain are themselves evidence of limitations in managing pain. In 2011, the Institute of Medicine released a report entitled, *Relieving Pain in America: A Blueprint for Transforming Prevention, Care, Education, and Research*, that outlined disparities between the impact of pain and current investments in all domains listed in their subtitle.[4] It addressed reasons for these disparities as well as possible solutions. This article outlines the current evidence for chronic pain

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as a disease, taxonomies of pain, and an approach to the assessment of painful conditions. Subsequent articles in this issue delve in greater depth into these topics individually.

THE EXPERIENCE OF PAIN

Pain is defined as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage."[5] This definition indicates several things about pain. First, it combines a sensory perception with an unpleasant emotional experience. These are distinct aspects of pain that reflect distinct pathways of activation of somatosensory and limbic cerebral cortex.[6] These 2 pathways can be uncoupled in pathology. In pain asymbolia, a rarely described syndrome that can be a consequence of injury to insular cortex, patients accurately describe nociceptive stimulation as painful but do not perceive it as unpleasant. Furthermore, the attention-motivational component of the pain experience, which results in withdrawal from painful stimuli and protection of the affected area, is absent in pain asymbolia, indicating that the responsible lesion interrupts both the emotional and motivational aspects of the experience of pain.[7] Second, pain descriptors are commonly words that describe tissue damage. This seems appropriate, because physiologic pain is initiated by stimulation of nociceptors, which are transducing elements on nerve terminals that only initiate an action potential in the context of a stimulus capable of inducing tissue damage. Finally, this definition specifies that pain is an experience and, therefore, is both subjective and does not have an obligate correspondence with nociceptive stimulation. Pain can only be measured as it is reported.

That said, the report of pain as a symptom may be a surrogate for a wide variety of concerns, only one of which is a desire for pain relief. People who present with pain as a chief complaint may be motivated to seek help because of fear, anxiety, loss of life roles, disrupted sleep, functional and occupational disability, dependency, addiction, or other factors. Even if not explicitly acknowledged as goals of therapy separate from pain relief itself, these state, trait, and situational factors can influence reported pain severity itself.

THE BIOLOGY OF CHRONIC PAIN

There is a growing body of evidence that chronic pain can exist as a disease state. This concept is supported by both clinical and preclinical evidence. In the clinical realm, several distinct pain syndromes have been demonstrated to present with a consistent symptom complex, no evident end-organ pathology, and reproducible differences from healthy control subjects in psychophysical, neurophysiological, or functional neuroimaging studies.[8,9] This literature suggests that these conditions are disorders of somatosensory and pain signaling in the nervous system. Preclinical evidence in animal models of chronic inflammation or nerve injury provide robust models of peripheral and central sensitization modulated by second messenger systems and epigenetic modifications responding to repetitive nociceptor stimulation.[10] This supports the view that acute pain conditions can, in certain circumstances, lead to sensitization that persists well after acute tissue injury has resolved or exacerbates pain perception in the context of chronic nociceptor stimulation. These data are further buttressed by identification of genetic and epigenetic factors that may predispose to sensitization of nociceptive pathways in the nervous system.[11,12]